simplex virus, and confluent measles keratitis with or without exposure.[2,3]

In the rural areas of developing countries, microbiological and biochemical investigations cannot be carried out rapidly and accurately. Therefore, history and morphological appearance of the corneal ulceration become important in the diagnosis and treatment. Measles infection must be kept in mind in the differential diagnosis of such an appearance as in our patients, especially in children from endemic countries. If it is detected in time, it can be treated with vitamin A therapy, antibiotic eye drops, and eye patching.

In conclusion, post-measles blindness can be prevented by generalizing the vaccination programmes and by the addition of adequate vitamin A in the diet of preschool children in endemic regions of the world.

References

1 Dekkers NWHM. Post measles blindness. *Doc Ophthalmol* 1983; **56**: 137–141.
2 Foster A, Sommer A. Corneal ulceration, measles, and childhood blindness in Tanzania. *Br J Ophthalmol* 1987; **71**: 331–343.
3 Sommer A. Nutritional factors in corneal xerophthalmia and keratomalacia. *Arch Ophthalmol* 1982; **100**: 399–403.
4 Sandford-Smith JH, Whittle HC. Corneal ulceration following measles in Nigerian children. *Br J Ophthalmol* 1979; **63**: 720–724.
5 Rapoza PA, West SK, Katala SJ, Munoz B, Taylor HR. Etiology of corneal opacification in central Tanzania. *Int Ophthalmol* 1993; **17**: 47–51.

M Köksal, Ş Kargi and S Uğurbaş

Zonguldak Karaelmas University School of Medicine
Department of Ophthalmology
Turkey

Correspondence: M Köksal,
Zonguldak Karaelmas Universitesi
Tıp Fakültesi Göz Hastalıkları AD
Zonguldak, Turkey
Tel: +90 372 2610169
Fax: +90 372 2610155
E-mail: mrtkksl@hotmail.com

Sir,

Intraocular cysticercosis simulating retinoblastoma in a 5-year-old child

Eye (2003) **17,** 447–449. doi:10.1038/sj.eye.6700340

Several ocular conditions can clinically simulate retinoblastoma by producing either a leucocoria or an ophthalmoscopically visible intraocular mass. In a large series of patients referred with the clinical diagnosis of retinoblastoma, 42% were found to have benign lesions that simulated retinoblastoma.[1] The three lesions that most often clinically mimic retinoblastoma are persistent hyperplastic primary vitreous, Coats' disease, and ocular toxocariasis.[1] Herein we report a case of intraocular cysticercosis in a 5-year-old child presenting with leucocoria, and simulating retinoblastoma.

Case report

A systemically healthy 5-year-old male child was referred with a history of pain and redness in the left eye (LE) of 20 days duration. He had been initially diagnosed and treated as a case of conjunctivitis, and later suspected to have endophthalmitis. On examination, the LE had no light perception. There was mild lid oedema, conjunctival congestion, and mild corneal oedema. The anterior chamber was deep with 2+flare and a trace of cells. The lens was clear and there was a white pupillary reflex. The intraocular pressure by Perkins applanation tonometer was unrecordably low. Indirect ophthalmoscopy revealed retinal detachment and a large ($20 \times 18 \times 16$ mm) intravitreal vascularized yellowish mass. A- and B-scan ultrasonography demonstrated an intraocular mass occupying most of the vitreous cavity and choroidal thickening (Figure 1). The mass showed several areas of high internal reflectivity and orbital shadowing (Figure 1), suggestive of intraocular calcification. As a result of inflammation associated with a calcified intraocular mass in a child, a necrotic retinoblastoma and a toxocara granuloma were considered in the differential diagnosis. The sightless and symptomatic eye was enucleated and sent for histopathologic examination.

On gross examination, the eye was normal in size. The cut section revealed a large solid intravitreal mass with an eccentrically situated cystic cavity. The microscopic examination showed total retinal detachment, solid vitreoretinal proliferation, diffuse infiltration by lymphocytes, plasma cells, eosinophils, and an eccentric abscess cavity (Figure 2). A cyst with a wall lined by a wavy cuticle and a smooth muscle layer was noted within the abscess cavity. Giant cell reaction was noted within the surrounding cyst wall (Figure 2, inset). The choroid was thickened with oedema, congested vessels, and mononuclear cell infiltration. Although definite scolex and hooklets were not identified, the characteristics of the cyst wall were diagnostic of cysticercosis.

Comment

Cysticercosis, an infestation by the larval form of the cestode *Taenia solium*, is a common ocular parasitosis.[2]

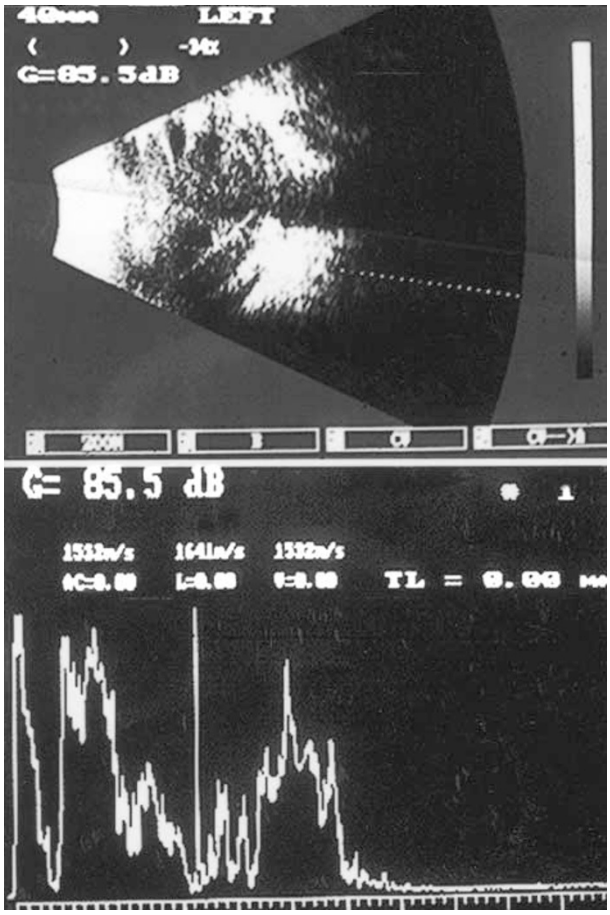

Figure 1 Ultrasonography B-scan (top) demonstrates an echodense intravitreal-space-occupying lesion with varying echogenecity, almost filling the entire vitreous cavity. Note the orbital shadowing. The corresponding vector A-scan (bottom) shows high internal reflectivity.

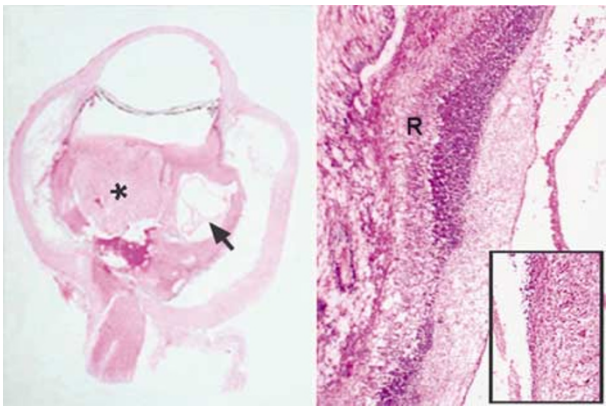

Figure 2 The whole mount section of the eyeball (left) shows a solid vitreoretinal mass (*), occupying the entire vitreous cavity. Note the abscess with a cyst (arrow) at the periphery of the mass (haematoxylin and eosin, $\times 5$). The microscopic examination of the section of the eyeball (right) shows degenerating wall of the cysticercus within the cavity, with multinucleated giant cells (inset) in the wall of abscess cavity (haematoxylin and eosin, $\times 250$).

Once considered as an endemic disease in developing countries, there has been a gradual change in the socio-demographic trends of ocular cysticercosis.[3] The disease is now being diagnosed with increasing frequency the world over, and is no longer a medical curiosity.[2,3] On review of all documented cases of ocular cysticercosis, it was found that 35% of the cysts were reported in the subretinal space, and 22% in the vitreous.[2] Intraocular cysticercosis usually presents with reduction of vision, and signs of ocular inflammation.[2–4] It is believed that the larva reaches the subretinal space through posterior ciliary arteries.[2–5] As the cyst develops, it may cause exudative retinal detachment.[4,5] Perforation of retina results in a free-floating intravitreous cyst.[2] Most often, the characteristic intraocular cyst can be visualized by an indirect ophthalmoscope, or detected by ultrasound B-scan in the presence of media haze.[2] Histologically, cysticercus consists of a single invaginated protoscolex within the cavity of fluid-filled cyst. The protoscolex has suckers and hooklets, while the three-layered cyst wall consists of a cuticle, a smooth muscle layer, and tegumental cells with scattered calcareous corpuscles.

As long as the cyst remains viable, it evokes a little or no inflammatory response. Once the cyst starts degenerating, an antigen, which may be a metabolic by-product or toxin, leaks from the cyst and induces an inflammatory reaction,[2] manifesting as vitritis, uveitis, and sometimes endophthalmitis.[2–5] An unusual feature of our patient was that he presented as leucocoria with ocular inflammatory signs, and an ultrasonographic appearance of calcification. This clinical presentation in a child strongly points towards the diagnosis of necrotic rentinoblastoma.[1,6] The density of the inflammatory reaction within the vitreoretinal mass surrounding the cyst probably hindered the detection of the cyst by ultrasound B-scan and simulated intraocular calcification. The degenerating parasite possibly evoked the surrounding granulomatous reaction and the vitreoretinal proliferation.

Although rare, intraocular cysticercosus could present as leucocoria and pose a diagnostic problem in children. It should be considered in the differential diagnosis of leucocoria presenting with ocular inflammation, especially in endemic areas.

References

1 Shields JA, Parsons HM, Shields CL, Shah P. Lesions simulating retinoblastoma. *J Pediatr Ophthalmol Strabismus* 1991; **28**: 338–340.

2 Kruger-Leite E, Jalkh AE, Quiroz H *et al*. Intraocular cysticercosis. *Am J Ophthalmol* 1985; **99**: 252–257.
3 Atul K, Kumar H, Mallika G, Sandip M. Socio-demographic trends in ocular cysticercosis. *Acta Ophthalmol Scand* 1995; **73**: 438–441.
4 Topilow HW, Yimoyines DJ, Freeman HM *et al*. Bilateral multifocal intraocular cysticercosis. *Ophthalmology* 1981; **88**: 1166–1172.
5 Bartholomew RS. Subretinal cysticercosis. *Am J Ophthalmol* 1975; **79**: 670–673.
6 Shields JA, Shields CL, Eagle RC *et al*. Calcified intraocular abscess simulating retinoblastoma. *Am J Ophthalmol* 1992; **114**: 227–229.

B Agarwal[1], GK Vemuganti[2] and SG Honavar[3]

[1]Department of Comprehensive Ophthalmology
LV Prasad Eye Institute
LV Prasad Marg
Banjara Hills
Hyderabad 500 034
India

[2]Ophthalmic Pathology Service
LV Prasad Eye Institute
LV Prasad Marg
Banjara Hills
Hyderabad 500 034
India

[3]Ocular Oncology Service
LV Prasad Eye Institute
LV Prasad Marg
Banjara Hills
Hyderabad 500 034
India
Correspondence: GK Vemuganti
Tel: +91 40 360 8262
Fax: +91 40 354 8271
E-mail: geeta@lvpeye.stph.net

Sir,

Scleromalacia as a complication of herpes zoster ophthalmicus

Eye (2003) **17,** 449–451. doi:10.1038/sj.eye.6700336

Reported scleral complications of herpes zoster ophthalmicus (HZO) include episcleritis, scleritis,[1–4] and scleromalacia.[5,6] Scleromalacia is an exceedingly rare complication of HZO and it can pose a diagnostic problem to the unwary ophthalmologist. Thinning and discolouration of the sclera may raise the question of an extrascleral extension of a uveal melanoma. In fact, enucleation of an affected eye was performed in one of the cases[7] with scleromalacia following HZO. All previous reported cases of scleromalacia following HZO were preceded by severe pain.[5–7] Scleromalacia following HZO was preceded by episodes of acute[5] or possibly chronic scleral inflammation. Scleritis and episcleritis can recur in subsequent years, resulting in scleral atrophy.[1] We describe a female patient who developed scleromalacia without a history of pain and precipitating scleritis, $1\frac{1}{2}$ years after being diagnosed with HZO.

Case report

A 79-year-old lady was referred by her GP for further evaluation of a black mass in her left eye. She complained of deteriorating vision in her left eye 2–3 months prior to this presentation. She gave no history of pain or inflammation of the eye. One and a half years before this presentation, she suffered an acute attack of severe left-sided HZO, complicated by a large noninfective epithelial defect, mild anterior uveitis (+1 cells) and raised intraocular pressure requiring admission. For a week, treatment included oral antiviral therapy, anti-glaucoma and mydriatic drops. A topical steroid was not used because by the time the epithelial defect healed, the anterior chamber activity had subsided. The outcome was an uneventful recovery without any further sequelae. Her general medical history was otherwise unremarkable.

On this presentation, unaided visual acuity was 6/6 in the right eye and 6/60 improving to 6/12 with a correction of $+0.25/+4.50 \times 180$. There was a left relative afferent pupillary defect and a well-defined, superotemporal, dark discolouration of the sclera, a few millimetres from the limbus (Figure 1), which could be brightly transilluminated. There was no associated iris

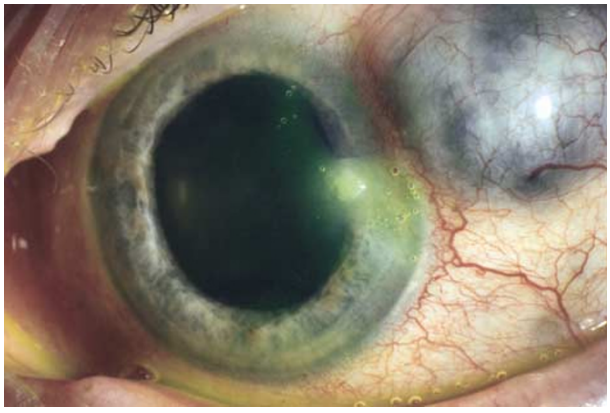

Figure 1 Anterior segment photograph of the left eye showing the superotemporal area of scleromalacia.